HEPATOLOGY INTRODUCTION & VIRAL HEPATITIS

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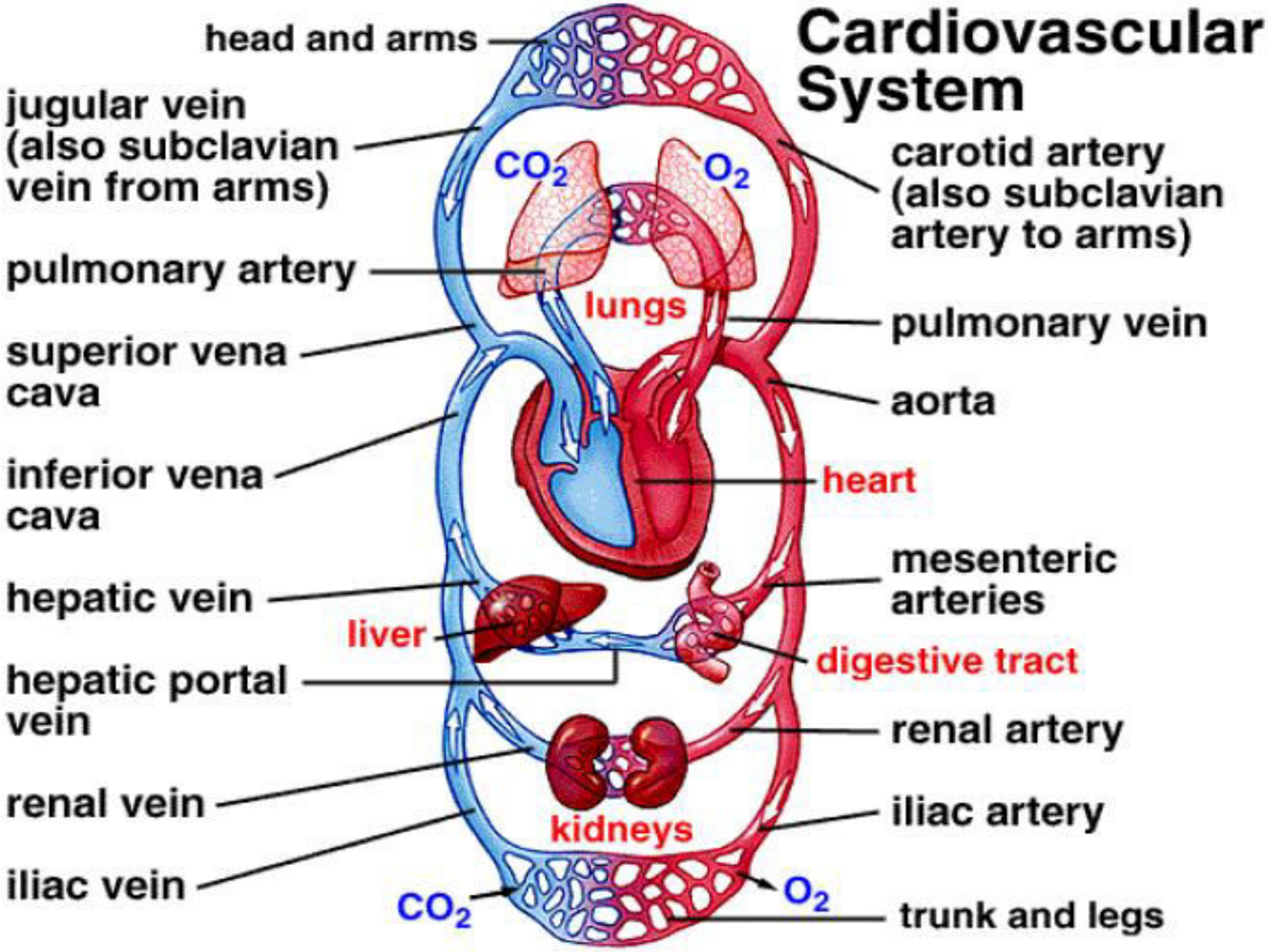
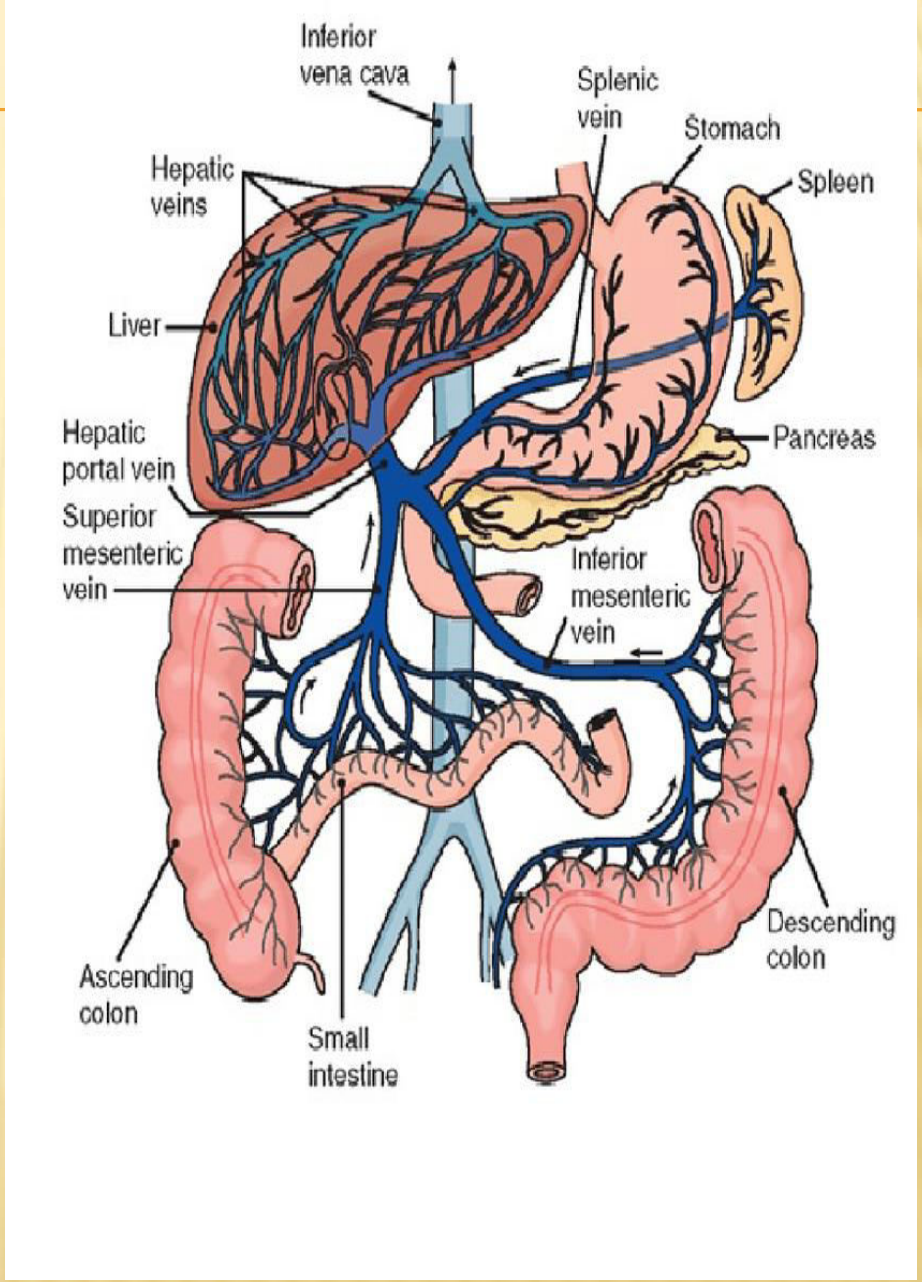
Cardiovascular System



INTRODUCTION 1

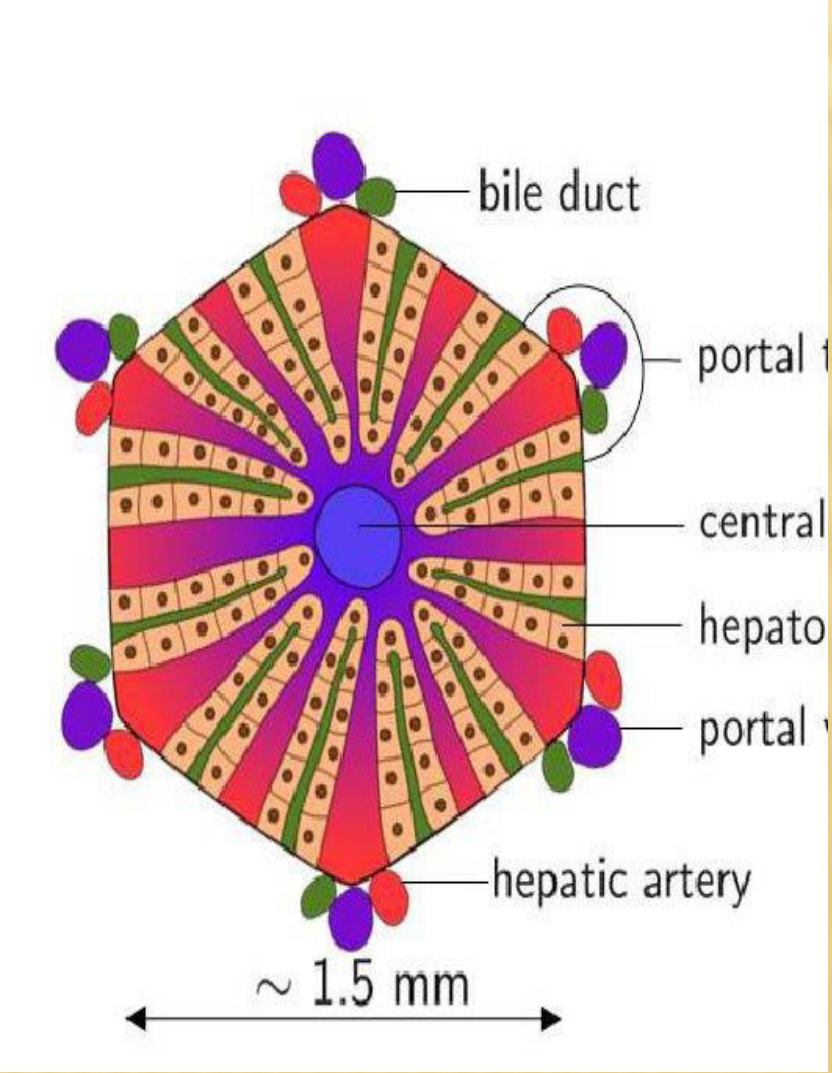
- The liver is largest organ in the body (1.2-1.5 kg) and is situated in the right hypochondrium.
- The hepatic blood supply is via two main vessels:

- The hepatic artery, a branch of the celiac axis, supplies 25% of the hepatic blood flow.
- The portal vein drains most of the gastrointestinal tract and the spleen. It supplies 75% of hepatic blood flow. The normal portal pressure is 5-8 mmHg.



INTRODUCTION 2

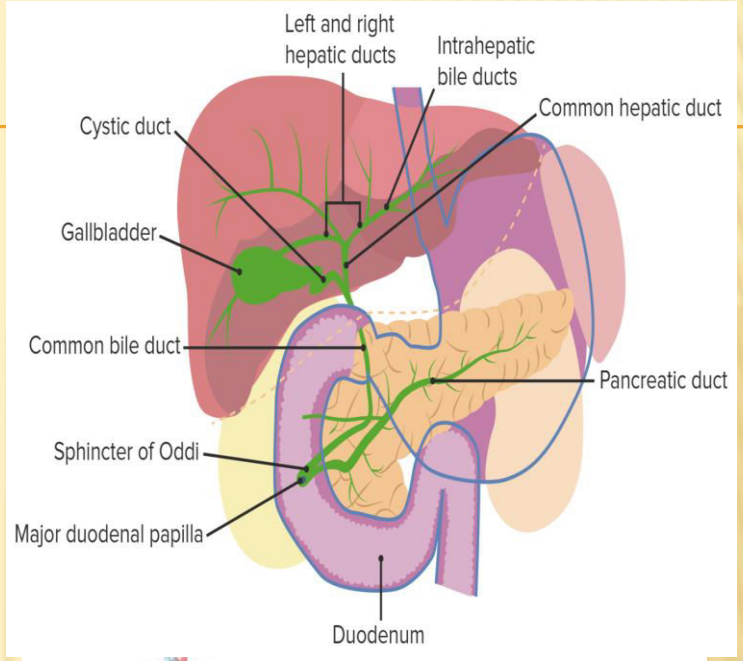
- The blood from these vessels is distributed to the segments and flows into the sinusoids via the portal tracts. Blood leaves the sinusoids, entering branches of the hepatic vein which join into three main branches before entering the inferior vena cava.
- **The hepatic lobule:** the hepatic lobule is the basic unit of the liver. In the centre, there is a central tributary of the hepatic vein and at the periphery, the portal tracts containing branches of the portal vein, hepatic artery and the bile duct. Cords of liver cells and sinusoids extend between these two systems. The portal tracts are surrounded by a limiting plate of liver cells.

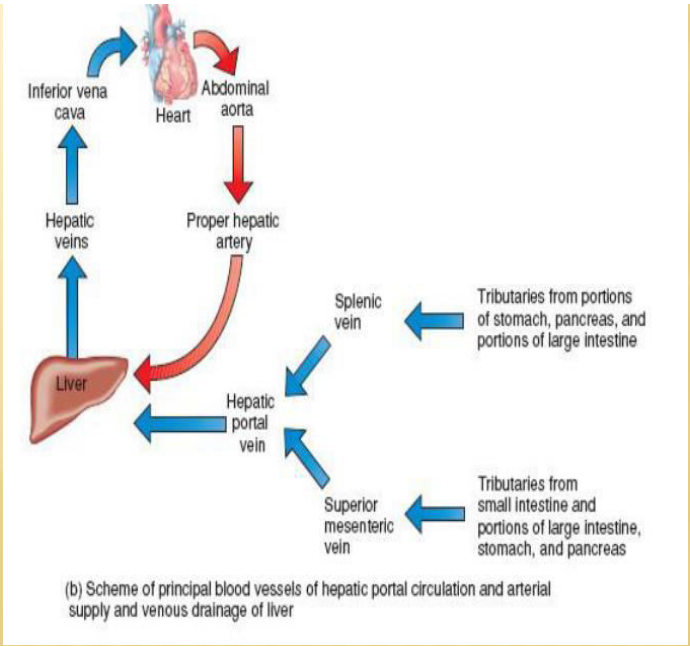


INTRODUCTION3

The biliary system:

- Bile canaliculi form a network between the hepatocytes. These join to form thin bile ductules → bile ducts in the portal tracts. → combine to form the right and left hepatic ducts. The hepatic ducts join at the porta hepatis to form the common hepatic duct.
- The gall bladder stores and concentrates hepatic bile; its capacity is approximately 50 ml. The common bile duct is formed at the junction of the cystic and common hepatic duct, passing through the head of the pancreas to pass into the duodenum. The common bile duct and pancreatic duct open into the second part of the duodenum most often through a common channel at the ampulla of Vater.





(b) Scheme of principal blood vessels of hepatic portal circulation and arterial supply and venous drainage of liver

ACUTE VIRAL HEPATITIS

ACUTE HEPATITIS VERSUS ACUTE VIRAL HEPATITIS 1

WHAT IS ACUTE HEPATITIS????

- Definition; Acute damage and inflammation of the liver cells can be caused by several agents:

Viral infections	•Viral hepatitis: A, B , D , C &E. • Epstein- Barr virus. •Cytomegalovirus.
Non viral infection	•Toxoplasmosis. •Leptospirosis. •Q fever.
Drugs	Paracetamol. Alcohol.
Poisons	Amanita phalloides (mushrooms).
Others	Pregnancy. circulatory insufficiency Wilson's disease .

WHAT IS ACUTE VIRAL HEPATITIS????

Definition:

- It is an acute damage & inflammation of the entire liver caused by viral infections affecting mainly the hepatic parenchyma.
- Viral hepatitis is responsible for 1-2 million deaths annually.

Etiology:

HAV, HBV, HCV, HDV and HEV

ACUTE HEPATITIS VERSUS ACUTE VIRAL HEPATITIS 2

CLINICAL PICTURE

1- Anicteric hepatitis:

- It may present with influenza like symptoms or gastroenteritis symptoms. No jaundica and patient is usually undiagnosed.

2- Icteric hepatitis:

a- Prodromal period (few days)

- The patient feels not well and complains of marked malaise, anorexia and nausea.
- Mild fever with pain in right upper abdomen
- Urine is dark and stools are slightly light

b- Icteric stage:

- Jaundice appears
- Fever drops and appetite and general condition improve
- The liver is enlarged, tender and soft
- Spleen may be enlarged

c- Convalescent stage: ▪ Start of recovery (rapid in children)

- Stools and urine regain their color and jaundice disappears
- Fatigue persists for some weeks.

INVESTIGATIONS

- **CBC:** WBCs shows leucopenia with relative lymphocytosis.
- **Viral markers:**
- Detection of Anti viral Abs IgM in acute infection or IgG in remote or chronic carrier state.
- Detection of viral DNA or RNA by PCR.
- **Liver function tests:**
- Marked elevation of Liver enzymes ALT and AST (500—5000 u/l).
- Serum bilirubin is increased (both direct and indirect).
- With liver failure: elevated INR, decreased Albumin.

SEQUELAE AND COMPLICATIONS 1

1. **Complete recovery** is the rule in almost all HAV& HEV infections, also in about 90% of HBV infections in adults. Only 20% of HCV patients recover spontaneously.
2. **Relapse or prolonged cholestasis:** Especially in HAV infections. In relapse The attack is usually milder attack, and is marked by elevation of transaminases and serum bilirubin. Recovery usually follows.

SEQUELAE AND COMPLICATIONS 2

3. **Acute liver cell failure** marked by deep jaundice and coagulopathy followed by confusion, coma due to brain oedema and death.
4. **Chronic persistant infection:**

- HBV carrier rate depends on age (95% in infants and only 5% in adults).
- HDV super-infection causes chronic hepatitis in about 70%.
- HCV infections persist in about 80%.

TREATMENT

- No specific treatment available.
- Rest : Bed rest is mandatory until the patient is free of jaundice & symptoms and the bilirubin is less than 1.5mg/dl.
- Diet: traditionally the patient was given a low fat, high CHO diet as it is said to be the most appetizing.
- Antiviral therapies for HBV & HCV patients may be used in acute hepatitis, to decrease incidence of developing chronic infection.
- Avoidance of hepatotoxic drugs.

1- HEPATITIS A VIRUS (HAV)

- is the most common viral hepatitis occurring worldwide.
- Often in epidemics. The disease is commonly seen in the autumn and affects children and young adults.
- Spread of infection is mainly by the feco-oral route and arises from the ingestion of contaminated food.
- HAV is a picornavirus, with an incubation period ranging between 2-6 weeks.
- No chronic infection in HAV.

- **markers**: An anti-HAV IgM means an acute infection , HAV IgG means old infection and immunity.
- The mortality in young adults is 0.1 % but it increases with age. Death is due to acute fulminant liver failure.
- Prophylaxis by improvement of sanitary (hygienic) conditions.
- Active immunization by formaldehyde-inactivated HAV vaccine is available and could be used for high risk children.

HEPATITIS B VIRUS (HBV) 1

- It is a DNA, hepadna virus
- Transmission:

1- Parenteral by transfusion of infected blood or use of contaminated needles or syringes.

2- Sexual intercourse.

3- From mother to baby.

- Incubation period: 2-6 months. The clinical attack may be mild or severe.
- Recovery occurs in about 95% of adults and only 5% of infants born to HBV positive mothers, and the rest of patients may pass through chronic hepatitis, liver cirrhosis and hepatocellular carcinoma.

HEPATITIS B VIRUS (HBV) 2

- Viral markers:

1- HBsAg: means ongoing infection - disappears after 3 months of infection - if persist more than 6 months, the patient is chronic carrier.

2-Anti-HBs:indicates recovery and immunity.

3- Anti-HBc: denotes exposure to the virus, current or past.

4- HBeAg: denotes high viral replication in an infected patient.

5- Anti HBe: denote low viral replication in an infected patient.

	HBsAg	Anti-HBs	Anti-HBc
Susceptible	Negative	Negative	Negative
Vaccinated	Negative	Positive	Negative
Past Infection	Negative	Positive	Positive
Acute Infection	Positive	Negative	IgM Positive
Chronic Infection	Positive	Negative	IgG Positive

HEPATITIS B VIRUS (HBV) 3

Vaccine is Recombinant vaccine (HBsAg) 3 doses 0-1-6 months

Passive Prophylaxis:

Immunoglobulins to acutely exposed persons and to infants born to HbsAg positive mother .

Active Prophylaxis:

Hepatitis B vaccine should be given to:

a- Surgical and dental stuff including students

b- Hospital staff in contact with blood

c- Babies born to HBsAg positive mother

d- Family and sexual contacts of HBsAg positive carriers

HEPATITIS C VIRUS (HCV)

- HCV is a single-stranded RNA of the flaviviridae family.
- HCV is highly endemic in Egypt & considered the main cause of liver cirrhosis and Hepatocellular carcinoma.
- Transmission occurs mainly after transfusion of contaminated blood or blood products and 3% by contaminated needle pricking.
- There is a low frequency of sexual and vertical transmission
- Incubation period: 2-6 months.
- Acute illness is usually mild and may be subclinical. However, about 80% of patients pass to chronic hepatitis, liver cirrhosis and hepatocellular carcinoma.
- Prophylaxis: Prevention of nosocomial infection and supervision of blood donation. No vaccine available.
- HCV markers:
  - HCV antibodies (by ELIZA) indicates current or past infection
  - HVC RNA: using PCR indicates active viral replication

HEPATITIS D VIRUS (HDV)

- It is an incomplete virus, requires HBV for infection
- Parental Transmission: drug abuse – blood born – sexual and rarely vertical.
- Infection may occur as:
  - Co-infection together with HBV
  - Superinfection in a chronic hepatitis B carriers.
- It may lead to chronic liver disease.
- Prophylaxis by vaccination against HBV.
- Incidence decrease globally due to HBV vaccination programs.

HEPATITIS E VIRUS (HEV)

- HEV is a RNA virus causing hepatitis clinically very similar to hepatitis A.
- Fecal– oral route transmission (mainly contaminated water).
- It has mortality from fulminant hepatic failure of 1-2%, which rises to 20% in pregnant women.
- There is no carrier state and it does not progress to chronic liver disease except in some immunosuppressed patients.
- An ELISA for IgG and IgM anti-HEV is available for diagnosis. HEV RNA can be detected in the serum or stools by PCR.
- Prevention and control depend on good sanitation and hygiene; a vaccine has been developed.

- Dental medical students & dentists are susceptible to infection with parental transmission of virus HBV, HCV and HDV through abrasion or cut wounds of the skin. They should receive HBV vaccine.
- Dentists may transmit the infection between patients & the instruments should be autoclaved and gloves should be changed between a patient and the next. Infection control parameters should be applied in dental clinics.

THANK YOU